Welcome to DIALOG

Dialog level 05.20.01D
%%%HILIGHT set on as '%%%'
? b 411;set files biotech
26oct07 12:56:06 User219511 Session D706.2
$0.00 0.115 DialUnits File410
$0.00 Estimated cost File410
$0.02 TELNET
$0.02 Estimated cost this search
$0.52 Estimated total session cost 0.259 DialUnits
File 411:DIALINDEX(R)

DIALINDEX(R)
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*** DIALINDEX search results display in an abbreviated ***
*** format unless you enter the SET DETAIL ON command. ***
You have 25 files in your file list.
(To see banners, use SHOW FILES command)
? s (IP-10 or IP10) and (pregnan? or conceptus or conception or embryo or uterin? or uterus or implant? or steril? or fertil?)

Your SELECT statement is:
s (IP-10 or IP10) and (pregnan? or conceptus or conception or embryo or uterin? or uterus or implant? or steril? or fertil?)

Items	File
14	5: Biosis Previews(R)_1926-2007/Oct W3
1	24: CSA Life Sciences Abstracts_1966-2007/Jun
17	34: SciSearch(R) Cited Ref Sci_1990-2007/Oct W3
3	71: ELSEVIER BIOBASE_1994-2007/Oct W2
18	135: NewsRx Weekly Reports_1995-2007/Oct W3
4	144: Pascal_1973-2007/Oct W2
2	155: MEDLINE(R)_1950-2007/Oct 23
1	172: EMBASE Alert_2007/Oct 19
8	357: Derwent Biotech Res.__1982-2007/Sep W5
10	399: CA SEARCH(R)_1967-2007/UD=14718

10 files have one or more items; file list includes 25 files.

? save temp; b 155,5,34,71,172,399;exs;rd
Temp SearchSave "TI48626289" stored
26oct07 12:57:42 User219511 Session D706.3
$5.07 1.724 DialUnits File411
$5.07 Estimated cost File411
$0.53 TELNET
$5.60 Estimated cost this search
$6.12 Estimated total session cost 1.982 DialUnits

SYSTEM:OS - DIALOG OneSearch
File 155:MEDLINE(R) 1950-2007/Oct 23
(c) format only 2007 Dialog
File 5:Biosis Previews(R) 1926-2007/Oct W3
(c) 2007 The Thomson Corporation
File 34:SciSearch(R) Cited Ref Sci 1990-2007/Oct W3
(c) 2007 The Thomson Corp
File 71:ELSEVIER BIOBASE 1994-2007/Oct W2
(c) 2007 Elsevier B.V.
File 172:EMBASE Alert 2007/Oct 19
(c) 2007 Elsevier B.V.
File 399:CA SEARCH(R) 1967-2007/UD=14718
(c) 2007 American Chemical Society
*File 399: Use is subject to the terms of your user/customer agreement.
IPCR/8 classification codes now searchable as IC=. See HELP NEWSIPCR.

Set	Items	Description
		Executing TI48626289
	1377	IP-10
	750	IP10
	1152324	PREGNAN?
	8365	CONCEPTUS
	48632	CONCEPTION
	573768	EMBRYO
	286033	UTERIN?
	206878	UTERUS
	686756	IMPLANT?
	229151	STERIL?
	850852	FERTIL?
S1	47	(IP-10 OR IP10) AND (PREGNAN? OR CONCEPTUS OR CONCEPTION OR EMBRYO OR UTERIN? OR UTERUS OR IMPLANT? OR STERIL? OR FERTIL?)
S2	36	RD (unique items)

? t s2/7/1-36;bye

2/7/1 (Item 1 from file: 155)
DIALOG(R)File 155:MEDLINE(R)
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22911164 PMID: 16839404
T-helper type 2 polarization among asthmatics during and following %%%pregnancy%%%.
Rastogi D; Wang C; Lendor C; Rothman P B; Miller R L
Division of Pulmonary, Allergy, Critical Care, Department of Medicine, College of Physicians and Surgeons, Columbia University, New York, NY 10032, USA.
Clinical and experimental allergy - journal of the British Society for Allergy and Clinical Immunology (England) Jul 2006, 36 (7) p892-8, ISSN 0954-7894--Print Journal Code: 8906443
Contract/Grant No.: 1 P01 AI50514; AI; NIAID; RR00645; RR; NCRR
Publishing Model Print
Document type: Journal Article; Multicenter Study; Research Support, N.I.H., Extramural
Languages: ENGLISH
Main Citation Owner: NLM
Record type: MEDLINE; Completed
BACKGROUND: Asthma is the most common medical condition during %%%pregnancy%%% . While increased production of T helper cytokines has been reported to occur in both asthma and %%%pregnancy%%% , the effect of T-helper type 2 (Th2) polarization on asthma symptoms during %%%pregnancy%%% has not been well-characterized. OBJECTIVE: We hypothesized that systemic Th2 cytokine and chemokine polarization occurs among asthmatics to a greater extent during their %%%pregnancy%%% , and is associated with more severe asthma and increased Th2 polarization in the newborn. METHODS: Fifty-six %%%pregnant%%% asthmatics were recruited from prenatal clinics affiliated with New York Presbyterian Hospital. Systemic production of interleukin-4, interferon-gamma, eotaxin and %%%IP10%%% were measured by intracytoplasmic staining or ELISA at recruitment, peripartum and post-partum, and in the cord blood. The frequency of asthma symptoms was measured by questionnaires and compared with Th biomarkers. RESULTS: The chemokine ratio (%%%IP10%%%/eotaxin) declined over the course of %%%pregnancy%%% (from 3.3 +/- 1.3 to 1.4 +/- 0.2, P = 0.016), but %%%IP10%%% and eotaxin increased post-partum. The decrease in the chemokine ratio was associated with more frequent asthma symptoms. A non-significant trend towards decreased interferon-gamma and increased interleukin-4 production was detected. Cord blood eotaxin levels correlated with maternal levels (r = 0.35, P = 0.03). Other peripartum biomarkers were not associated with Th2 polarization nor with subsequent respiratory symptoms in the newborn. CONCLUSION: %%%IP10%%%/eotaxin declined over the course of %%%pregnancy%%% and was associated with worse asthma symptoms. Alterations of Th1/Th2 chemokine balance during %%%pregnancy%%% may identify women prone to more severe asthma during %%%pregnancy%%%.
Record Date Created: 20060714
Record Date Completed: 20070222

2/7/2 (Item 2 from file: 155)
DIALOG(R)File 155:MEDLINE(R)

(c) format only 2007 Dialog. All rts. reserv.

13098662 PMID: 11219493
Adenovirus-mediated delivery of antiangiogenic genes as an antitumor approach.
Regulier E; Paul S; Marigliano M; Kintz J; Poitevin Y; Ledoux C; Roecklin D; Cauet G; Calenda V; Homann H E
TRANSGENE S.A., Strasbourg, France.
Cancer gene therapy (England) Jan 2001, 8 (1) p45-54, ISSN 0929-1903--Print Journal Code: 9432230
Publishing Model Print
Document type: Journal Article; Research Support, Non-U.S. Gov't
Languages: ENGLISH
Main Citation Owner: NLM
Record type: MEDLINE; Completed
Based on the observation that the growth of solid tumors is dependent on the formation of new blood vessels, therapeutic strategies aimed at inhibiting angiogenesis have been proposed. A number of proteins with angiostatic activity have been described, but their development as therapeutic agents has been hampered by difficulties in their production and their poor pharmacokinetics. These limitations may be resolved using a gene therapy approach whereby the genes are delivered and expressed in vivo. Here we compared adenoviral delivery of endostatin, proliferin-related protein (PRP), and interferon-inducible protein 10 (%%%IP10%%%) genes. Recombinant adenoviruses carrying the three angiostatic genes express biologically active gene products as determined in vitro in endothelial cell proliferation and migration assays, and in vivo by inhibition of neoangiogenesis in rat chambers. Eradication of established tumors in vivo, in the murine B16F10 melanoma model in immunocompetent mice, was not achieved by intratumoral injection of the different vectors. However, the combination of intravenous plus intratumoral injections allowed rejection of tumors. Ad-PRP or Ad-%%%IP10%%% were significantly more efficient than Ad-endostatin, leading to complete tumor rejection and prolonged survival in a high proportion of treated animals. These data support the use of in vivo gene delivery approaches to produce high-circulating and local levels of antiangiogenic agents for the therapy of local and metastatic human tumors.
Record Date Created: 20010221
Record Date Completed: 20010426

2/7/3 (Item 1 from file: 5)
DIALOG(R)File 5:Biosis Previews(R)
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0019770071 BIOSIS NO.: 200700429812
Expression of IP-10 related to angiogenesis in %%%uterine%%% cervical cancers
AUTHOR: Sato E; Fujimoto J (Reprint); Toyoki H; Sakaguchi H; Alam S M; Jahan I; Tamaya T
AUTHOR ADDRESS: Gifu Univ, Sch Med, Dept Obstet and Gynecol, 1-1 Yanagido, Gifu 5011194, Japan**Japan
AUTHOR E-MAIL ADDRESS: jf@gifu-u.ac.jp
JOURNAL: British Journal of Cancer 96 (11): p1735-1739 JUN 4 2007 2007
ITEM IDENTIFIER: doi:10.1038/sj.bjc.6603790
ISSN: 0007-0920_(print) 1532-1827_(electronic)
DOCUMENT TYPE: Article
RECORD TYPE: Abstract
LANGUAGE: English

ABSTRACT: Angiogenesis is essential for development, growth and advancement of solid tumours. Interferon-gamma-inducible protein 10 (IP-10) regulates lymphocyte chemotaxis, mediates vascular pericyte proliferation and acts as an angiostatic agent, thus inhibiting tumour growth. This prompted us to study the clinical implications of IP-10 expression related to angiogenesis in %%%uterine%%% cervical cancers. The levels of IP-10 decreased with advancement, and the prognosis of the 30 patients with low IP-10 expression in %%%uterine%%% cervical cancers was poor (66%), whereas the 24-month survival rate of the other patients with high IP-10 expression was 90%. Furthermore, IP-10 levels significantly reverse-correlated with vascular endothelial growth factor (VEGF) levels in %%%uterine%%% cervical cancers. Interferon-gamma-inducible protein 10 might work on suppression of angiogenesis associated with VEGF in advancement, and can be recognised as a prognostic indicator. Furthermore, IP-10 activation might be effective on the suppression of regrowth or recurrence after intensive treatment for advanced cervical cancers.

2/7/4 (Item 2 from file: 5)
DIALOG(R)File 5:Biosis Previews(R)
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0019679226 BIOSIS NO.: 200700338967
27th Annual Meeting of the American-Society-for-Reproductive-Immunology, Toronto, CANADA, May 14 -16, 2007
AUTHOR: Anonymous
JOURNAL: American Journal of Reproductive Immunology 57 (5): p308-352 MAY 2007 2007
CONFERENCE/MEETING: 27th Annual Meeting of the American-Society-for-Reproductive-Immunology Toronto, CANADA May 14 -16, 2007; 20070514
ISSN: 1046-7408
DOCUMENT TYPE: Meeting; Meeting Summary
RECORD TYPE: Abstract
LANGUAGE: English

ABSTRACT: This meeting, which focuses on reproductive immunology, contains 94 abstracts written in English. Topics include the male and female reproductive tract, reproductive tract infections, reproductive cancer and reproductive failure.

2/7/5 (Item 3 from file: 5)
DIALOG(R)File 5:Biosis Previews(R)
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0019664289 BIOSIS NO.: 200700324030
Role of the chemokine receptor CCR5 in the clearance and development of immunity against Chlamydia trachomatis
AUTHOR: Barr E L (Reprint); Okwandu E; Thurman S L; McMillan L; Ifere G; He Q; Eko E; Igietseme J U; Ananaba G A
AUTHOR ADDRESS: Clark Atlanta Univ, Atlanta, GA 30314 USA**USA
JOURNAL: Abstracts of the General Meeting of the American Society for Microbiology 104 p217-218 2004 2004
CONFERENCE/MEETING: 104th General Meeting of the American-Society-for-Microbiology New Orleans, LA, USA May 23 -27, 2004; 20040523
SPONSOR: Amer Soc Microbiol
ISSN: 1060-2011
DOCUMENT TYPE: Meeting; Meeting Abstract
RECORD TYPE: Citation
LANGUAGE: English

2/7/6 (Item 4 from file: 5)
DIALOG(R)File 5:Biosis Previews(R)
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0019561953 BIOSIS NO.: 200700221694
Is CXCL10/%%%IP10%%% the missing link between inflammation and anti-angiogenesis in preeclampsia?
AUTHOR: Gotsch Francesca (Reprint); Friel Lara; Kusanovic Juan Pedro; Espinoza Jimmy; Erez Offer; Than Nandor Gabor; Mittal Pooja; Edwin Samuel ; Yoon Bo Hyun; Mazaki-Tovi Shali; Hassan Sonia; Romero Roberto
AUTHOR ADDRESS: NICHHD, Natl Inst Hlth, DHHS, Perinatol Res Branch, Detroit, MI USA**USA
JOURNAL: American Journal of Obstetrics and Gynecology 195 (6, Suppl. S): pS153 DEC 2006 2006
CONFERENCE/MEETING: 27th Annual Meeting of the

Society-of-Maternal-Fetal-Medicine San Francisco, CA, USA February 05 -10, 2007; 20070205
SPONSOR: Soc Maternal Fetal Med
ISSN: 0002-9378
DOCUMENT TYPE: Meeting; Meeting Abstract
RECORD TYPE: Citation
LANGUAGE: English

2/7/7 (Item 5 from file: 5)
DIALOG(R)File 5:Biosis Previews(R)

18707492 BIOSIS NO.: 200600052887
A mouse model for Ad37 corneal infection
AUTHOR: Chintakuntlawar A (Reprint); Astley R A; Xiao J; Rajala M S; Callegan M C; Chodosh J
JOURNAL: IOVS 46 (Suppl. S): p1019 2005 2005
CONFERENCE/MEETING: Annual Meeting of the Association-for-Research-in-Vision-and-Ophthalmology Ft Lauderdale, FL, USA May 01 -05, 2005; 20050501
SPONSOR: Assoc Res Vis & Ophthalmol
ISSN: 0146-0404
DOCUMENT TYPE: Meeting; Meeting Poster
RECORD TYPE: Abstract
LANGUAGE: English

ABSTRACT: Purpose: Adenoviruses are major human pathogens and a common cause of ocular surface infection. Subgroup D adenoviruses, including Ad8, Ad19, and Ad37, are responsible for significant ocular morbidity as the etiologic agents of epidemic keratoconjunctivitis. We have previously shown in vitro that the earliest host responses to adenovirus infection of keratocytes are controlled by intracellular signaling cascades that activate within minutes of exposure to the virus and result in the expression of chemokines. However, in vivo confirmation ofintracellular signaling as the pathogenic mechanism is lacking. A mouse modelwould offer obvious advantages over in vitro tissue culture models, but humanadenoviruses do not replicate in murine cells. We sought to create a mouse model of adenovirus-induced inflammatory gene expression that would not require adenoviral replication. Methods: Intracorneal injection of cesium chloride gradient-purified Ad37 was performed in 6-8 week old female Balb/c mice using %%%sterile%%% glass needles and a CO2 powered injection system. Corneas wereharvested 4 hrs post-injection, and homogenized in TRIzol for RNA isolation. Total tissue RNA was subjected to quantitative real-time PCR for analysis of mRNA expression for select inflammatory mediators. Untouched, needle only, and dialysis buffer-injected corneas were used as controls. Results: Histology of injected corneas demonstrated intrastromal corneal blebs without perforation of Descemets membrane. Real-time PCR analysis of the proinflammatory mediators KC, IP-10, and IL-6 demonstrated on average a 3-5 fold higher expression of mRNA at 4 hrs post-injection in the Ad37-injected corneas compared to buffer-injected corneas. Conclusions: Our approach successfully delivered Ad37 to the corneal stroma of BALB/c mice, and induced reproducible increases in gene expression for several important mediators of inflammation. The injection technique used should allow analysis of the role of host cell signaling even in the absence of adenoviral replication.

2/7/8 (Item 6 from file: 5)
DIALOG(R)File 5:Biosis Previews(R)

18631644 BIOSIS NO.: 200510326144
Hormonal regulation of TLR3-induced responses
AUTHOR: Lesmeister Margaret J (Reprint); Jorgenson Rebecca L; Young Steven L; Misfeldt Michael L
AUTHOR ADDRESS: Univ Missouri, Columbia, MO 65212 USA**USA
JOURNAL: FASEB Journal 19 (4, Suppl. S, Part 1): pA948 MAR 4 2005 2005
CONFERENCE/MEETING: Experimental Biology 2005 Meeting/35th International Congress of Physiological Sciences San Diego, CA, USA March 31 -April 06, 2005; 20050331
SPONSOR: Amer Assoc Anatomists
Amer Assoc Immunologists
Amer Physiol Soc
Amer Soc Biochem & Mol Biol
Amer Soc Investigat Pathol
Amer Soc Nutr Sci
Amer Soc Pharmacol & Expt Therapeut
Int Union Physiol Sci
ISSN: 0892-6638
DOCUMENT TYPE: Meeting; Meeting Abstract
RECORD TYPE: Abstract
LANGUAGE: English

ABSTRACT: Cyclic expression of TLR3 mRNA and protein in endometrial tissues and up-regulation of TLR3 during the %%%implantation%%% window have been observed in our laboratory. We have demonstrated TLR3 expression in the endometrial epithelial cell lines RL95-2 and HEC-1-A. TLR3 stimulation with the synthetic ligand, polyinosinic:polycytidylic acid (poly I:C), results in activation of the transcription factors nuclear factor kappa B (NF-kappa B) and interferon regulatory factor 3(IRF3), with subsequent production of cytokines and chemokines involved in endometrial function and regulation. Cycle-dependent expression of TLR3 suggestspossible modulation of TLR3 function by the endometrial hormones, estradiol (E) and progesterone (P). We have observed that RL95-2 cells express estrogen receptor (ER) alpha, ER beta, and progesterone receptor. Pretreatment of poly I:C-stimulated RL95-2 cells with E results in a reduction of IL-6, IL-8, and IP-10 production as compared to controls. P had no effect. The effects of E are ER-dependent, as shown using ER antagonists and a TLR3-positive, ER-negative cell line, HEC-1-A. E treatment does not affect TLR3 mRNA or protein expression. Current studies are focused on elucidating the mechanisms by which E modulates TLR3 function. NIH R21 A155504-02.

2/7/9 (Item 7 from file: 5)
DIALOG(R)File 5:Biosis Previews(R)

18254414 BIOSIS NO.: 200500160586
Changes in immune cell distribution and IL-10 production are regulated through endometrial IP-10 expression in the goat %%%uterus%%%
AUTHOR: Imakawa Kazuhiko (Reprint); Nagaoka Kentaro; Nojima Hisashi; Hara Yasuhiro; Christenson Ronald K
AUTHOR ADDRESS: Fac AgrLab Anim BreedingImplantat Res Grp,Bunkyo Ku, Univ Tokyo, 1-1-1 Yayoi, Tokyo, 1138657, Japan**Japan
AUTHOR E-MAIL ADDRESS: akaz@mail.ecc.u-tokyo.ac.jp
JOURNAL: American Journal of Reproductive Immunology 53 (1): p54-64 January 2005 2005
MEDIUM: print
ISSN: 1046-7408 _(ISSN print)
DOCUMENT TYPE: Article
RECORD TYPE: Abstract
LANGUAGE: English

ABSTRACT: PROBLEM: Changes in distribution or redistribution of immune cells are required for the establishment and maintenance of %%%pregnancy%%%, but these changes during early %%%pregnancy%%% have been poorly understood in the ruminant ungulates. Expression of a chemokine, interferon-gamma (IFN-gamma)-inducible protein 10 kDa (IP-10, CXCL10), was identified in the endometrium of %%%pregnant%%% goats. Population and/or distribution of endometrial immune cells and their cytokine productions could be regulated by IP-10 during the period of %%%pregnancy%%% establishment. METHOD OF STUDY: Using reverse transcriptase-polymerase chain reaction (RT-PCR), expression of IP-10, IFN-gamma, tumor necrosis factor-alpha, interleukin-10 (IL-10), CXCR3 mRNA and leukocyte cell surface markers, CD4, CD8, CD11b and CD45 mRNA during the caprine early %%%pregnancy%%% was investigated. The ability of IP-10 to stimulate peripheral blood mononuclear cells (PBMCs) migration

was demonstrated using a chemotaxis assay. Changes in migration of PBMCs' immune cell population and cytokine expressions with IP-10 stimulation were investigated using flow cytometry and RT-PCR respectively. RESULTS: Levels of IP-10, IL-10, CD4 and CD11b mRNA, and the number of CD4 and CD11b positive cells in %%%pregnant%%% goat endometrium were higher than those of cyclic goat endometrium. Migration of PBMCs was stimulated by recombinant caprine IP-10, and the effect was significantly reduced by neutralization with the use of an anti-IP-10 antibody. In the flow cytometric and RT-PCR analyses, migrated cells stimulated by IP-10 increased the expression of IL-10 and CD11b mRNA. Furthermore, IP-10 could stimulate the expression of IL-10 mRNA from PBMCs. CONCLUSION: Endometrial chemokine IP-10 could regulate IL-10 production by resident and possibly migrated cells expressing CD11b, probably natural killer cells, and these changes may result in immune environments of the %%%uterus%%% suitable for %%%conceptus%%% %%%implantation%%% in ruminants.

2/7/10 (Item 8 from file: 5)
DIALOG(R)File 5:Biosis Previews(R)

18243861 BIOSIS NO.: 200500150926
Identification of 2,3,7,8-tetrachlorodibenzo-p-dioxin (TCDD)-inducible and -suppressive genes in the rat placenta: Induction of interferon-regulated genes with possible inhibitory roles for angiogenesis in the placenta
AUTHOR: Mizutani Tetsuya; Yoshino Miki; Satake Tomoko; Nakagawa Miyuki; Ishimura Ryuta; Tohyama Chiharu; Kokame Koichi; Kangawa Kenji; Miyamoto Kaoru (Reprint)
AUTHOR ADDRESS: Fac Med SciDept Biochem, Fukui Univ, Matsuoka, Fukui, 9101193, Japan**Japan
JOURNAL: Endocrine Journal 51 (6): p569-577 December 2004 2004
MEDIUM: print
ISSN: 0918-8959
DOCUMENT TYPE: Article
RECORD TYPE: Abstract
LANGUAGE: English

ABSTRACT: Exposure to a low dose of 2,3,7,8-tetrachlorodibenzo-p-dioxin (TCDD) results in a variety of toxic manifestations, including fetal death. In order to evaluate the effects of low dose TCDD on placental function, %%%pregnant%%% Holtzman rats were given a single oral dose of 1600 ng TCDD/kg body wt or an equivalent volume of vehicle (control) on gestation day 15 (GD15), and changes in the gene expression in the placenta on GD20 were analyzed by two comprehensive methods, representational difference analysis (RDA) and DNA microarray technology. Candidates of TCDD-inducible and -suppressive genes were selected. Quantitative real-time PCR analysis was then performed to verify the induction or suppression levels of the candidate genes. Finally, we identified 81 TCDD-inducible and 21 TCDD-suppressive genes from the placenta of TCDD-treated Holtzman rats on GD20. One of the remarkable profiles of the gene expression was that glucose transporters were strongly up-regulated by the TCDD treatment. Furthermore, many interferon-inducible genes were also up-regulated by the treatment. They included several cytokines Such as IP-10 known as a potent angiogenesis inhibitor. In addition, interferon molecules are known to suppress angiogenesis. The above observations suggest that activation of the interferon signaling pathway and the induction of anti-angiogenic factors by TCDD might have a role in causing the inhibition of neovascularization, resulting in the hypoxic state of placenta and increased incidence of fetal death.

2/7/11 (Item 9 from file: 5)
DIALOG(R)File 5:Biosis Previews(R)

17654646 BIOSIS NO.: 200400025403
Effects of chemokines and chemokine receptors in the regulation of anti-Chlamydia specific immunity.
AUTHOR: Barr E L (Reprint); Okwandu E (Reprint); Bennett W (Reprint); Ifere G; Ramey K; McMillan L; Moore T; Stewart J S (Reprint); Igietseme J U; Ananaba G A (Reprint)
AUTHOR ADDRESS: Clark Atlanta Univ., Atlanta, GA, USA**USA
JOURNAL: Abstracts of the Interscience Conference on Antimicrobial Agents and Chemotherapy 43 p64 2003 2003
MEDIUM: print
CONFERENCE/MEETING: 43rd Annual Interscience Conference on Antimicrobial Agents and Chemotherapy Chicago, IL, USA September 14-17, 2003; 20030914
SPONSOR: American Society for Microbiology
DOCUMENT TYPE: Meeting; Meeting Abstract
RECORD TYPE: Abstract
LANGUAGE: English

ABSTRACT: Background: Chlamydia trachomatis is the most widespread cause of sexually transmitted diseases such as pelvic inflammatory disease, ectopic %%%pregnancy%%% and infertility in the United States. Animal models and human studies have shown that the immune response against Chlamydia involves the secretion of chemokines and cytokines such as IFN-gamma and IL-12 by susceptible cells. The purpose of this study is to elucidate the role of chemokines and chemokine receptors in the induction of protective immunity against Chlamydia which could be relevant in vaccine development. We hypothesized that chemokines as well as their receptors play a vital role in the induction of anti-chlamydia specific immunity. Methods: In this study we infected CCR5 (Th1 chemokine receptor) knockout mice intravaginally with C. trachomatis (106 IFU/ml). Harvested genital tracts (GT), spleens, lymph nodes and the supernatants from nylon wool isolated T-cells were assessed by ELISA for the presence of IFN-gamma and IL-10 cytokines and RANTES, IP-10 and MCP1 chemokines. Results: Anti-Chlamydia specific Th1 response was detected 7 days post infection. No measurable Th1 response was detected 14 days post infection, and there was no Th2 response detected at any time. In addition, we observed a preferential expression of CCR5, over CCR3 (Th2 chemokine receptor) in Chlamydia infected genital tract tissues. Moreover, systemic T-cell response was totally eliminated in chemokine receptor knockout animals. Conclusion: These results suggest that the lack of a chemokine receptor influences the outcome of Chlamydia infection by abrogating retention of T-cells in the GT, reducing IFN-gamma production, and decreasing production of anti-Chlamydia agents.

2/7/12 (Item 10 from file: 5)
DIALOG(R)File 5:Biosis Previews(R)

17564429 BIOSIS NO.: 200300519792
Influence of chemokines and chemokine receptors in the pathogenesis and complications of Chlamydia infection.
AUTHOR: Ananaba G A (Reprint); Okwandu E O (Reprint); Ogunkoya Y (Reprint); Barr E (Reprint); Eko F O; Moore T; Ramey K; McMillan L; Stewart J (Reprint); Igietseme J U
AUTHOR ADDRESS: Clark Atlanta University, Atlanta, GA, USA**USA
JOURNAL: Abstracts of the General Meeting of the American Society for Microbiology 103 pB-038 2003 2003
MEDIUM: cd-rom
CONFERENCE/MEETING: 103rd American Society for Microbiology General Meeting Washington, DC, USA May 18-22, 2003; 20030518
SPONSOR: American Society for Microbiology
ISSN: 1060-2011 _(ISSN print)
DOCUMENT TYPE: Meeting; Meeting Abstract
RECORD TYPE: Abstract
LANGUAGE: English

ABSTRACT: The potential threat of genital chlamydia infection to human reproduction, well-being and national healthcare budget is of great concern because in women a Chlamydia induced cervical disease may ascend into the upper genital tract, and cause serious complications, such as pelvic inflammatory disease (PID), fallopian tube scarring, ectopic %%%pregnancy%%% and infertility. The molecular basis for the susceptibility of certain individuals in the population to the sequelae

of Chlamydia infection is unclear. Obvious limitations in human experimentation have led to the development of several animal models with a goal to understanding the pathogenesis and immunobiology of the chlamydial disease. In humans as well as animals, Th1 cells are mostly responsible for genital chlamydia clearance. It has been shown also that the dynamics of cytokines, chemokines and their receptors elicited during Chlamydia infection influence immune response leading to protection or pathology. We tested the hypothesis that the differential susceptibility of individuals to Chlamydia is influenced by the profile of cytokines, chemokines and chemokines receptors expressed during Chlamydia infection. Using different strains of mice and by semi-quantitative PCR analysis, we measured the levels of chemokine and chemokine receptors in the genital tract of infected and uninfected animals. Nylon wool purified T cells from these animals were stimulated with inactivated antigen plus APCs, and 5 days later the supernatant was immunoassayed for cytokine secretion. The results showed differential expression of RANTES, MCP1, %%%IP10%%%, CCR5, CXCR3, CXCR1, CXCR2 and CXCR4 by these animals. In the two strains of mice studied the time course of chlamydia clearance correlated with the time of maximun expression of the chemokines and chemokine receptors. In addition, the levels of IFN-gamma and IL4 did not differ significantly in both animals. These results show that the genetic background of an individual can influence the person's susceptibility to Chlamydia infection depending on the chemokines and cytokines that are expressed.

2/7/13 (Item 11 from file: 5)
DIALOG(R)File 5:Biosis Previews(R)

17548700 BIOSIS NO.: 200300503728
Genetic deletion of chemokine receptor CXCR3 or antibody blockade of its ligand IP-10 modulates posttransplantation graft-site lymphocytic infiltrates and prolongs functional graft survival in pancreatic islet allograft recipients.
AUTHOR: Baker Marshall S; Chen Xiaojuan; Rotramel Alizah R; Nelson Jeffrey J; Lu Bao; Gerard Craig; Kanwar Yashpal; Kaufman Dixon B (Reprint)
AUTHOR ADDRESS: The Feinberg School of Medicine, Division of Organ Transplantation, Northwestern University, 675 N St. Clair St, Galter Pavilion, Suite 17-200, Chicago, IL, 60611, USA**USA
JOURNAL: Surgery (St Louis) 134 (2): p126-133 August 2003 2003
MEDIUM: print
ISSN: 0039-6060 _(ISSN print)
DOCUMENT TYPE: Article
RECORD TYPE: Abstract
LANGUAGE: English

ABSTRACT: Background. Interaction of chemokine receptor CXCR3 with its ligand IP-10 mediates effector cell trafficking to sites of allograft rejection in murine models of whole organ allotransplantation. We hypothesized that blocking the CXCR3/IP-10 interaction would impair posttransplantation leukocyte trafficking to and delay rejection of pancreatic islet allografts. Methods. A/J strain murine islets were %%%implanted%%% to the kidney capsule of H-2 disparate, streptozotocin induced diabetic wild type (WT), CXCR3 deficient (CXCR3-/-) or IP-10 antibody-treated WT (alphaIP-10) C57BL/6 recipients. Representative grafts from each group were harvested at day 7. Ribonuclease protection assay was used to determine gene expression for cell markers F4/80 (macrophages), CD8 (type I T cells), CD4 (type II T cells), and CD 19 (natural killer cells), and for chemokines IP-10, MIP-1alpha, MIP-1beta, MCP-1, and RANTES. Immunohistochemistry was used to confirm ribonuclease protection assay infiltrate data. Graft-site chemokine gene expression and cellular infiltrate were correlated with time to functional graft rejection. Results. Untreated WT recipients demonstrated heavy graft-site cell infiltrates and increased graft-site gene expression for cell markers F4/80, CD8, CD4, and CD19, and for chemokines RANTES, IP-10, and MIP-1beta at day 7. In comparison with untreated WT, alphaIP-10-treated WT and CXCR3-/- recipient demonstrated the same degree of chemokine gene expression but less lymphocytic infiltrate. The mean length of allograft survival was 12.7 +- 3.1 days in untreated WT versus 20.2 +- 2.7 days (P < .05) for CXCR3-/-- and 19.7 +- 2.3 days (P < .05) for alphaIP-10-treated WT recipients. Conclusions. CXCR3 gene deletion or alphaIP-10 antibody therapy modulates posttransplantation lymphocytic graft infiltration and statistically prolongs graft survival in murine islet allograft recipients.

2/7/14 (Item 12 from file: 5)
DIALOG(R)File 5:Biosis Previews(R)

17380521 BIOSIS NO.: 200300337264
Identification of Intermediary Steps in the SLC (CCL21) Chemokine Mediated Anti-Tumor Immune Response.
AUTHOR: Tolba Khaled A (Reprint); Bowers William J (Reprint); Muller Jacquelyn (Reprint); Giuliano Rita E (Reprint); Federoff Howard J (Reprint); Rosenblatt Joseph D (Reprint)
AUTHOR ADDRESS: James P. Wilmot Cancer Center, University of Rochester, Rochester, NY, USA**USA
JOURNAL: Blood 100 (11): pAbstract No. 2566 November 16, 2002 2002
MEDIUM: print
CONFERENCE/MEETING: 44th Annual Meeting of the American Society of Hematology Philadelphia, PA, USA December 06-10, 2002; 20021206
SPONSOR: American Society of Hematology
ISSN: 0006-4971
DOCUMENT TYPE: Meeting; Meeting Abstract
RECORD TYPE: Abstract
LANGUAGE: English

ABSTRACT: Chemokines are a large family of small proteins that coordinate leukocyte traffic between various lymphoid compartments and peripheral tissues, as well as cellular interaction of various immune effector cells within lymphoid tissues, SLC plays a central role in organizing lymph node structure, and mediating dendritic cell (DC):T cell interaction through shared expression of the chemokine receptor CCR7 on these cells. We reasoned that local elaboration of SLC can lead to co-localization of dendritic cells and T-cells and facilitate T-cell priming that ordinarily takes place within the T cell zone of the lymph node, within the tumor bed leading to the generation of a systemic anti-tumor immune response. Using HSV amplicon vectors to deliver SLC alone, or in combination with CD40L, we tested for the ability to eradicate established tumors in both the murine A20 B-cell lymphoma and the CT26 adeno-carcinoma models. Administration of HSV amplicon vectors encoding SLC (HSV-SLC) into previously established subcutaneous A20 and CT26 tumors resulted in heavy infiltration with CD4+, CD8+ T-cells and DCs. This correlated with complete tumor regression in the majority of mice bearing A20 tumors and a sharply reduced growth rate in the CT26 model relative to non-injected or HSV-lac injected tumors. Combined transduction with HSV-SLC and HSV-CD40L generated more potent anti-tumor activity than that seen with either vector alone in both tumor models. . In mice %%%implanted%%% bilaterally with A20 tumors and unilaterally transduced with HSV amplicons, higher rates of regression were seen using combined HSV-CD40L and HSV-SLC transduction compared to either vector delivered alone. Mice with regressing tumors developed long-term immunity when re-challenged with parental tumors. Splenocytes from mice that eradicated their tumors exhibited CTL activity following in-vitro priming. T-cell subset depletion studies revealed that CD8+ but not CD4+ T-cells were needed for the generation of the anti-tumor immune response. We sought to characterize the down-stream events leading to tumor regression using RT-PCR for a panel of chemokines/cytokines elaborated by recruited cells. We found that early on (2-3 days) following amplicon delivery, a panel of inflammatory chemokines (RANTES, MIP-1b, MDC) is released. Subsequent events (Day 6-8 post-transduction) in regressing tumors, included expression of mRNA for the p40 subunit of IL-12, g-interferon and IP-10. These data shows that SLC-mediated immune rejection of tumors is a multi-step process involving coordinate expression of inflammatory and constitutive chemokines elaborated in a sequential manner by various effector cells. It also suggests the HSV-SLC amplicon as a useful agent to study contribution of the innate and adaptive immune responses towards the generation of anti-tumor immune response.

2/7/15 (Item 13 from file: 5)
DIALOG(R)File 5:Biosis Previews(R)

16661826 BIOSIS NO.: 200200255337
Vascular endothelial growth factor induces IP-10 chemokine expression
AUTHOR: Lin Ching-Shwun (Reprint); Lin Guiting; Chen Kuo-Chiang; Ho Hao-Chung; Lue Tom F
AUTHOR ADDRESS: Knuppe Molecular Urology Laboratory, Department of Urology, School of Medic, University of Califomia at San Francisco, San Francisco, CA, 94143-1695, USA**USA
JOURNAL: Biochemical and Biophysical Research Communications 292 (1): p 79-82 March 22, 2002 2002
MEDIUM: print
ISSN: 0006-291X
DOCUMENT TYPE: Article
RECORD TYPE: Abstract
LANGUAGE: English

ABSTRACT: We have previously shown that intracavernous injection of vascular endothelial growth factor (VEGF) improved the recovery of erectile function in an arteriogenic impotence rate mode. We wished to identify genes that are affected by VEGF treatment in the penis. Specifically we examined the induction of IP-10 chemokine. Male rats were subjected to pudendal arterial ligation or sham operation. They were then treated with intracavernous injection of 4 mug of VEGF in phosphate-buffered saline (PBS) or PBS alone. At 6 and 24 h after treatment, the erectile tissue was then harvested for RNA isolation. The isolated RNA was used for microarray and RT-PCR analyses. Cultured rat cavernous smooth muscle cells (CSMC) were treated with VEGF and then subjected to RT-PCR analysis. Cultured human CSMC were treated with VEGF and then subjected to ELISA analysis. Microarray analysis detected IP-10 as an abundantly induced message in 6-h VEGF-treated tissues. This was further confirmed by RT-PCR analysis. Using cultured rat CSMC, induction of IP-10 mRNA was detectable in 1 and 2 h, but not 24 h, VEGF-treated cells. Induction of IP-10 at the protein level was observed with cultured human CSMC. Secretion of IP-10 into culture medium peaked at 4 h after treatment of human CSMC with 10 ng/ml of VEGF. Optimal VEGF dosage for IP-10 induction was 50 ng/ml when assayed with cells that were treated for 8 h.

2/7/16 (Item 1 from file: 34)
DIALOG(R)File 34:SciSearch(R) Cited Ref Sci

14707855 Genuine Article#: 997TY Number of References: 56
Title: Toll-like receptor 3 agonist poly(I : C)-induced antiviral response in human corneal epithelia[cells
Author(s): Kumar A; Zhang J; Yu FSX (REPRINT)
Corporate Source: Wayne State Univ,Sch Med, Kresge Eye Inst,4717 St Antoine/Detroit//MI/48201 (REPRINT); Wayne State Univ,Sch Med, Kresge Eye Inst,Detroit//MI/48201(fyu@med.wayne.edu)
Journal: IMMUNOLOGY, 2006, V117, N1 (JAN), P11-21
ISSN: 0019-2805 Publication date: 20060100
Publisher: BLACKWELL PUBLISHING, 9600 GARSINGTON RD, OXFORD OX4 2DQ, OXON, ENGLAND
Language: English Document Type: ARTICLE
Abstract: The objective of this study was to examine the expression of Toll-like receptor 3 (TLR3) by human corneal epithelial cells (HCECs) and to determine whether exposure to the TLR3 agonist polyinosinic-polycytidylic acid [poly(I:C)] induces an antiviral response in these cells. Fluorescence-activated cell sorter (FACS) analysis revealed TLR3 to be constitutively expressed and distributed intracellularly in HCECs. Stimulation of HCECs with the TLR3 agonist poly(I:C) induced the activation of nuclear factor (NF)-kappa B and production of the proinflammatory cytokine interleukin (IL)-6 and the chemokine IL-8. Upon exposure to poly(I:C), HCECs initiated a potent antiviral response resulting in an increase of interferon (IFN)-beta mRNA expression (7-fold). Poly(I:C) stimulation also up-regulated mRNA expression of the antiviral chemokine IFN-gamma inducible protein 10 (%%%IP10%%%), myxovirus resistance gene A and 2',5'-oligoadenylate synthetase (5-, 10- and 9-fold, respectively), and secretion of %%%IP10%%%. These responses were also induced by exogenously added type 1 IFNs, but could not be blocked by pretreatment of the cells with anti-TLR3 monoclonal antibody, suggesting that the receptor was not expressed on the cell surface. Furthermore, incubation of HCECs with an endosomal acidification inhibitor, chloroquine, markedly inhibited poly(I:C)-mediated IFN-beta expression in HCECs. These results suggest that corneal epithelial cells are important sentinels of the corneal innate immune system against viral infection, and that stimulation of TLR3 can induce the expression of key proinflammatory cytokines and chemokines and antiviral genes that help in the defence of the cornea against viral infection.

2/7/17 (Item 2 from file: 34)
DIALOG(R)File 34:SciSearch(R) Cited Ref Sci

13370452 Genuine Article#: 872GH Number of References: 98
Title: Human immunodeficiency virus type 1 Nef protein mediates neural cell death: a neurotoxic role for IP-10
Author(s): van Marle G; Henry S; Todoruk T; Sullivan A; Silva C; Rourke SB; Holden J; McArthur JC; Gill MJ; Power C (REPRINT)
Corporate Source: Univ Calgary,Neurosci Res Grp, Dept Clin Neurosci,3330 Hosp Dr NW,HMRB 150/Calgary/AB T2N 4N1/Canada/ (REPRINT); Univ Calgary,Neurosci Res Grp, Dept Clin Neurosci,Calgary/AB T2N 4N1/Canada/ ; Univ Calgary,Dept Microbiol & Infect Dis,Calgary/AB T2N 4N1/Canada/; Univ Toronto,St Michaels Hosp, Dept Psychiat,Toronto/ON M5B 1W8/Canada/ ; Univ British Columbia,Dept Pathol,Vancouver/BC V5Z 1M9/Canada/; Johns Hopkins Univ,Dept Neurol,Baltimore//MD/21218; Johns Hopkins Univ,Dept Epidemiol,Baltimore//MD/21218(power@ucalgary.ca)
Journal: VIROLOGY, 2004, V329, N2 (NOV 24), P302-318
ISSN: 0042-6822 Publication date: 20041124
Publisher: ACADEMIC PRESS INC ELSEVIER SCIENCE, 525 B ST, STE 1900, SAN DIEGO, CA 92101-4495 USA
Language: English Document Type: ARTICLE
Abstract: HIV-1 Nef is expressed in astrocytes, but a contribution to neuropathogenesis and the development of HIV-associated dementia (HAD) remains uncertain. To determine the neuropathogenic actions of the HIV-1 Nef protein, the brain-derived (YU-2) and blood-derived (NL4-3) Nef proteins were expressed in neural cells using an alphavirus vector, which resulted in astrocyte death ($P < 0.001$). Supernatants from Nef-expressing astrocytes also caused neuronal death, suggesting the release of neurotoxic molecules by astrocytes. Analysis of pro-inflammatory gene induction in astrocytes expressing Nef revealed increased IP-10 mRNA expression (4000-fold) that was Nef sequence dependent. Recombinant IP-10 caused selective cell death in neurons ($P < 0.001$) but not astrocytes, and the cytotoxicity of supernatant from astrocytes expressing Nef YU-2 was blocked by an antibody directed against the chemokine receptor CXCR3 ($P < 0.001$). SCID/NOD mice %%%implanted%%% with a Nef YU-2-expressing vector displayed abnormal motor behavior ($P < 0.05$), neuroinflammation, and neuronal loss relative to controls. Analysis of mRNA levels in brains from patients with HAD also revealed increased expression of IP-10 ($P < 0.05$), which was confirmed by immunoreactivity detected principally in astrocytes. Phylogenetic and protein structure analyses of Nef sequences derived from HIV/AIDS patients with and without HAD suggested viral evolution toward a neurotropic Nef protein. These results indicate that HIV-1 Nef contributes to neuropathogenesis by directly causing astrocyte death together with indirect neuronal death through the cytotoxic actions of IP-10 on neurons. Furthermore, Nef molecular diversity was evident in brain tissue among patients with neurological disease and which may influence IP-10 production by astrocytes. (C) 2004 Elsevier Inc. All rights reserved.

2/7/18 (Item 3 from file: 34)
DIALOG(R)File 34:SciSearch(R) Cited Ref Sci
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11887871 Genuine Article#: 705XC Number of References: 44
Title: Regulation of blastocyst migration, apposition, and initial adhesion by a chemokine, interferon gamma-inducible protein 10 kDa (IP-10), during early gestation
Author(s): Nagaoka K; Nojima H; Watanabe F; Chang KT; Christenson RK; Sakai S; Imakawa K (REPRINT)
Corporate Source: Univ Tokyo,Fac Agr, Lab Anim Breeding, Bunkyo Ku,1-1-1 Yayoi/Tokyo 1138657//Japan/ (REPRINT); Univ Tokyo,Fac Agr, Lab Anim Breeding, Bunkyo Ku,Tokyo 1138657//Japan/; Higeta Shoyu Co Ltd,R&D Dept,Chiba 2888680//Japan/; Korea Res Inst Biosci & Biotechnol,Taejon 305333//South Korea/; ARS,Meat Anim Res Ctr, USDA,Clay Ctr//NE/68933
Journal: JOURNAL OF BIOLOGICAL CHEMISTRY, 2003, V278, N31 (AUG 1), P 29048-29056
ISSN: 0021-9258 Publication date: 20030801
Publisher: AMER SOC BIOCHEMISTRY MOLECULAR BIOLOGY INC, 9650 ROCKVILLE PIKE, BETHESDA, MD 20814-3996 USA
Language: English Document Type: ARTICLE
Abstract: For a %%%pregnancy%%% to be established, initial apposition and adhesion of the blastocyst to maternal endometrium must occur in a coordinated manner; however, a key factor(s) that mediates the trophoblast cell migration and attachment to the apical surface of the endometrium has not been identified. In this study, we examined the effect of an endometrial chemokine, interferon-gamma inducible protein 10 kDa (IP-10), on %%%conceptus%%% migration to the endometrial epithelium. We first studied endometrial IP-10 mRNA expression, which was localized in the subepithelial stromal region, and detected the protein in the %%%uterine%%% flushing media during early %%%pregnancy%%%. Expression of IP-10 mRNA by the endometrium of cyclic animals was stimulated by the addition of a %%%conceptus%%% factor interferon-tau (IFN-tau). Immunofluorescent analysis revealed that IP-10 receptor, CXCR3, was localized in the trophoblast cells, to which biotinylated-recombinant caprine IP-10 (rcIP-10) bound. Chemotaxis assay indicated that rcIP-10 stimulated the migration of trophoblast cells, and the effects of rcIP-10 were neutralized by the pretreatment with an anti-IP-10 antibody. Adhesive activity of trophoblast cells to fibronectin was promoted by rcIP-10, and the effect was inhibited by the use of anti-IP-10 antibody. Further adhesion experiments demonstrated that binding of trophoblast cells to fibronectin was completely inhibited by a peptide of the Arg-Gly-Asp(RGD) sequence, which binds to integrins alpha(5)beta(1), alpha(V)beta(1), alpha(V)beta(3), and alpha(V)beta(5), whereas non-binding peptide containing Arg-Gly-Glu(RGE) had minimal effects. More importantly, rcIP-10 promoted the adhesion of trophoblast cells to primary cells isolated from endometrial epithelium. Furthermore, rcIP-10 stimulated the expression of integrin alpha(5), alpha(V), and beta(3) subunit mRNA in trophoblast cells. These findings suggest that endometrial IP-10 regulates the establishment of apical interactions between trophoblast and epithelial cells during early gestation.

2/7/19 (Item 4 from file: 34)
DIALOG(R)File 34:SciSearch(R) Cited Ref Sci
(c) 2007 The Thomson Corp. All rts. reserv.

11783538 Genuine Article#: 695VZ Number of References: 28
Title: Detection of interferon-gamma-inducible chemokines in human milk
Author(s): Takahata Y (REPRINT) ; Takada H; Nomura A; Nakayama H; Ohshima K ; Hara T
Corporate Source: Kyushu Univ,Grad Sch Med Sci, Dept Paediat, Higashi Ku,3-1-1 Maidashi/Fukuoka 8128582//Japan/ (REPRINT); Kyushu Univ,Grad Sch Med Sci, Dept Paediat, Higashi Ku,Fukuoka 8128582//Japan/; Fukuoka Univ,Dept Pathol 1, Sch Med,Fukuoka 81401//Japan/
Journal: ACTA PAEDIATRICA, 2003, V92, N6 (JUN), P659-665
ISSN: 0803-5253 Publication date: 20030600
Publisher: TAYLOR & FRANCIS AS, CORT ADELERSGT 17, PO BOX 2562, SOLLI, 0202 OSLO, NORWAY
Language: English Document Type: ARTICLE
Abstract: Aim: To assess the immunological role of human milk by analysing the concentrations of interferon-gamma-inducible protein of 10 kda (IP-10) and monokine induced by interferon-gamma (MIG) in human milk from mothers of preterm and term infants. Methods: IP-10 and MIG levels of colostrum. early milk, mature milk and sera were measured by enzyme-linked immunosorbent assay (ELISA). IP-10 and MIG mRNA expression levels in cellular components of human milk were determined by RT-PCR. IP-10 and MIG protein expression in mammary gland tissues was analysed by immunohistochemistry. Results: Significant amounts of IP-10 and MIG were detected in human milk. The concentrations of IP-10 and MIG in colostrum and early milk were significantly higher than those of sera from healthy controls or lactating mothers. These chemokine concentrations in colostrum and early milk were significantly higher than those of mature milk. Premature delivery or %%%pregnancy%%% complications of mothers had no significant correlation with these chemokine concentrations in breast milk. There were significant correlations between MIG and interferon-gamma (IFN-gamma) or IP-10 levels ($p < 0.001$) in human milk. Expression of IP-10 and MIG genes and proteins in the milk cells as well as in mammary gland epithelial tissues was detected by RT-PCR and immunohistochemistry.

Conclusion: IP-10 and MIG in human milk, probably derived from milk cells and mammary gland epithelial cells, may contribute to the migration and activation of intestinal T lymphocytes to enhance mucosal immunity during the early neonatal period.

2/7/20 (Item 5 from file: 34)
DIALOG(R)File 34:SciSearch(R) Cited Ref Sci
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11395589 Genuine Article#: 645VF Number of References: 15
Title: Mumps virus decreases testosterone production and gamma interferon-induced protein 10 secretion by human Leydig cells
Author(s): Le Goffic R; Mouchel T; Ruffault A; Patard JJ; Jegou B; Samson M (REPRINT)
Corporate Source: Univ Rennes 1,GERM, INSERM, U435,Campus Beaulieu/F-35042 Rennes/Bretagne/France/ (REPRINT); Univ Rennes 1,GERM, INSERM, U435,F-35042 Rennes/Bretagne/France/; CHU Ponchaillou,Serv Bacteriol Virol,F-35000 Rennes//France/; CHU Ponchaillou,Serv Urol,F-35000 Rennes//France/
Journal: JOURNAL OF VIROLOGY, 2003, V77, N5 (MAR), P3297-3300
ISSN: 0022-538X Publication date: 20030300
Publisher: AMER SOC MICROBIOLOGY, 1752 N ST NW, WASHINGTON, DC 20036-2904 USA
Language: English Document Type: ARTICLE
Abstract: Mumps virus is responsible for %%%sterility%%%. Here, we show that the mumps virus infects Leydig cells in vitro and totally inhibits testosterone secretion and that ribavirin in mumps virus-infected Leydig cell cultures completely restores testosterone production. Moreover, we show that gamma interferon-induced protein 10 (IP-10) is highly expressed by mumps virus-infected Leydig cells and that ribavirin does not block IP-10 production.

2/7/21 (Item 6 from file: 34)
DIALOG(R)File 34:SciSearch(R) Cited Ref Sci
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10592714 Genuine Article#: 545QK Number of References: 56
Title: Suppression of metastatic hemangiosarcoma by a parvovirus MVMp vector transducing the IP-10 chemokine into immunocompetent mice
Author(s): Giese NA (REPRINT) ; Raykov Z; DeMartino L; Vecchi A; Sozzani S; Dinsart C; Cornelis JJ; Rommelaere J
Corporate Source: Deutsch Krebsforschungszentrum,Appl Tumor Virol Program F0100,D-69120 Heidelberg//Germany/ (REPRINT); Deutsch Krebsforschungszentrum,Appl Tumor Virol Program F0100,D-69120 Heidelberg//Germany/; Deutsch Krebsforschungszentrum,INSERM, U375,D-69120 Heidelberg//Germany/; IRF Mario Negri,Lab Inflammat &

Signal Transduct,I-20157 Milan//Italy/
Journal: CANCER GENE THERAPY, 2002, V9, N5 (MAY), P432-442
ISSN: 0929-1903 Publication date: 20020500
Publisher: NATURE PUBLISHING GROUP, MACMILLAN BUILDING, 4 CRINAN ST, LONDON N1 9XW, ENGLAND
Language: English Document Type: ARTICLE
Abstract: We have previously shown that the growth of human tumor xenografts in immunodeficient mice can be efficiently Suppressed upon infection with the autonomous parvovirus H-1 or with cytokine-transducing derivatives thereof. To further evaluate the benefits of implementing parvoviruses in cancer gene therapy, we have created a new recombinant vector, MVMp/IP-10, transducing the immunoactive, antiangiogenic chemokine IP-10, and used this virus to treat syngeneic tumors grown in immunocompetent mice. Intratumoral/intraperitoneal administration of only 3x 10(7) replication units of MVMp/IP-10 per animal strongly inhibited the progression of established H5V cell-induced vascular tumors? a highly malignant mouse model for human cavernous hemangioma and Kaposi's sarcoma. Retardation of recurrent tumor growth and suppression of life-threatening metastatic dissemination to internal organs were accompanied by a striking delay in hemangioma-associated mortality. Parental MVMp did not have a significant effect Under these conditions up to the dose of 10(10) infectious units/animal, but had strong antihemangiosarcoma activity when used to infect H5V cells ex vivo prior to %%%implantation%%%. In all cases, virus therapy was very well tolerated. Virus-induced suppression of hemangiosarcoma was dependent on host T cells and associated with intratumoral persistence of IFN-gamma-expressing cytotoxic lymphocytes, and led to the reduced expression of hepatic plasminogen activator inhibitor-1 (PAI-1), a metastasis-linked marker. This proof of principle study demonstrates that NAVMp/IP-10 can aid the treatment Of vascular tumors and that autonomous parvovirus-based vectors can be considered potent tools for cancer gene therapy purposes.

2/7/22 (Item 7 from file: 34)
DIALOG(R)File 34:SciSearch(R) Cited Ref Sci
(c) 2007 The Thomson Corp. All rts. reserv.

05713049 Genuine Article#: WR745 Number of References: 34
Title: Bovine granulocyte chemotactic protein-2 is secreted by the endometrium in response to interferon-tau (IFN-tau)
Author(s): Teixeira MG; Austin KJ; Perry DJ; Dooley VD; Johnson GA; Francis BR; Hansen TR (REPRINT)
Corporate Source: UNIV WYOMING,DEPT ANIM SCI, REPROD BIOL PROGRAM/LARAMIE//WY/82071 (REPRINT); UNIV WYOMING,DEPT ANIM SCI, REPROD BIOL PROGRAM/LARAMIE//WY/82071; UNIV WYOMING,DEPT MOL BIOL/LARAMIE//WY/82071; UNIV WYOMING,SCH PHARM/LARAMIE//WY/82071; NORTHWEST COLL,DEPT EQUINE SCI//POWELL//WY/
Journal: ENDOCRINE, 1997, V6, N1 (FEB), P31-37
ISSN: 0969-711X Publication date: 19970200
Publisher: HUMANA PRESS INC, 999 RIVERVIEW DRIVE SUITE 208, TOTOWA, NJ 07512
Language: English Document Type: ARTICLE
Abstract: Interferon-tau (IFN-tau) is secreted by the bovine %%%conceptus%%% and may regulate synthesis of %%%uterine%%% endometrial cytokines to provide an environment that is conducive to %%%embryo%%% development and %%%implantation%%%. Interferon-tau stimulates secretion of an 8-kDa %%%uterine%%% protein (P8) in the cow. P8 was purified, digested to yield internal peptides, and partially sequenced to determine identity. Two internal peptides had 100% (13-mer) and 92% (12-mer) amino acid sequence identity with bovine granulocyte chemotactic protein-2 (bGCP-2). Bovine GCP-2 is an alpha-chemokine that acts primarily as a potent chemoattractant for granulocyte cells of the immune system. A peptide was synthesized based on a region of bGCP-2 that overlapped with a P8 peptide amino acid sequence, coupled to keyhole limpet hemocyanin, and used to generate high titer polyclonal antiserum in sheep. Western blots revealed that bGCP-2 was not released by endometrium from day 14 nonpregnant cows, but was released in response to 25 nM IFN-tau ($p < 0.05$). %%%Uterine%%% GCP-2 exhibited high affinity to heparin agarose, a characteristic shared by all alpha chemokines. This is the first report describing presence of GCP-2 in the %%%uterine%%% endometrium and regulation by IFN-tau. The regulation of bGCP-2 by IFN-tau may have important implications for cytokine networking in the %%%uterus%%% during %%%pregnancy%%%. Also, the regulation of inflammation and angiogenesis by bGCP-2 working together with other cytokines may be integral to establishing early %%%pregnancy%%% and %%%implantation%%% in the cow.

2/7/23 (Item 8 from file: 34)
DIALOG(R)File 34:SciSearch(R) Cited Ref Sci
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05213066 Genuine Article#: VJ442 Number of References: 40
Title: INTERFERON-INDUCIBLE PROTEIN-10 INVOLVES VASCULAR SMOOTH-MUSCLE CELL-MIGRATION, PROLIFERATION, AND INFLAMMATORY RESPONSE
Author(s): WANG XK; YUE TL; OHLSTEIN EH; SUNG CP; FEUERSTEIN GZ
Corporate Source: SMITHKLINE BEECHAM PHARMACEUT,DEPT CARDIOVASC PHARMACOL,709 SWEDELAND RD,POB 1539,UW 2511/KING OF PRUSSIA//PA/19406
Journal: JOURNAL OF BIOLOGICAL CHEMISTRY, 1996, V271, N39 (SEP 27), P 24286-24293
ISSN: 0021-9258
Language: ENGLISH Document Type: ARTICLE
Abstract: Interferon-inducible protein-10 (IP-10) is a member of the C-X-C chemokine family. Using mRNA differential display, we isolated a rat homologue to murine and human IP-10 from lipopolysaccharide-stimulated carotid arteries. Our studies demonstrated that IP-10 is a potent mitogenic and chemotactic factor for vascular smooth muscle cells, the critical features of smooth muscle cells for their contribution to the pathogenesis of atherosclerosis and restenosis. IP-10 induced a concentration-dependent stimulation of DNA synthesis, cell proliferation, and cell migration of rat aortic smooth muscle cells. A concentration- and time-dependent IP-10 mRNA induction was observed in lipopolysaccharide- or interferon-gamma-stimulated, but not interleukin-1 beta- or tumor necrosis factor-alpha-stimulated smooth muscle cells. A marked synergistic effect on IP-10 mRNA expression was observed when smooth muscle cells were challenged with interferon-gamma together with interleukin-1 beta or tumor necrosis factor-alpha. Furthermore, IP-10 mRNA expression was induced in the rat carotid artery after balloon angioplasty. The mitogenic and chemotactic features of IP-10 for smooth muscle cells, along with its discrete induction in cultured vascular smooth muscle cells and in carotid arteries after balloon angioplasty (neointima formation) suggest that IP-10 may play an active and distinct role in vascular remodeling processes.

2/7/24 (Item 9 from file: 34)
DIALOG(R)File 34:SciSearch(R) Cited Ref Sci
(c) 2007 The Thomson Corp. All rts. reserv.

05050397 Genuine Article#: TL818 Number of References: 30
Title: MOB-1 EXPRESSION IN IL-2-INDUCED ARDS - REGULATION BY TNF-ALPHA
Author(s): NEVILLE LF; ABDULLAH F; MCDONNELL PM; YOUNG PR; FEUERSTEIN GZ; RABINOVICI R
Corporate Source: THOMAS JEFFERSON UNIV,JEFFERSON MED COLL,DEPT SURG,1025 WALNUT ST/PHILADELPHIA//PA/19107; SMITHKLINE BEECHAM PHARMACEUT,DEPT MOLEC IMMUNOL/KING OF PRUSSIA//PA/19406; SMITHKLINE BEECHAM PHARMACEUT,DEPT CARDIOVASC SURG/KING OF PRUSSIA//PA/19406
Journal: AMERICAN JOURNAL OF PHYSIOLOGY-LUNG CELLULAR AND MOLECULAR PHYSIOLOGY, 1995, V13, N6 (DEC), PL884-L890
ISSN: 1040-0605
Language: ENGLISH Document Type: ARTICLE
Abstract: We have recently established an animal model of adult respiratory distress syndrome (ARDS)-like microvascular lung injury elicited by infusion of human interleukin-2 (IL-2). Based on the pronounced, transcriptional upregulation of multiple pro-inflammatory mediators in IL-2-indueed ARDS, differential display was applied to search for potentially novel genes in this paradigm of lung injury. Differential

display on total lung RNA derived from IL-2-challenged rats presented a highly reproducible 3'-UTR fragment profile in which a band (approximate to 250 bp), termed B1, was strongly induced. B1 cDNA sequence exhibited 99.14% homology to the 3'-UTR of mob-1, a recently cloned gene belonging to the C-X-C chemokine superfamily. Furthermore, Northern blot analysis showed that IL-2-induced pulmonary mob-1 mRNA was expressed at time points before the onset of lung injury and suppressed after TNF-alpha inhibition. These data imply that lung mob-1 is a novel, highly inducible gene in a clinically relevant model of ARDS and, based on its identification as a chemokine, could participate in the development of lung injury.

2/7/25 (Item 10 from file: 34)
DIALOG(R)File 34:SciSearch(R) Cited Ref Sci

02655481 Genuine Article#: LU309 Number of References: 45
Title: IP-10, A -C-X-C- CHEMOKINE, ELICITS A POTENT THYMUS-DEPENDENT ANTITUMOR RESPONSE IN-VIVO
Author(s): LUSTER AD; LEDER P
Corporate Source: HARVARD UNIV,SCH MED,HOWARD HUGHES MED INST,DEPT GENET,200 LONGWOOD AVE/BOSTON//MA/02115; HARVARD UNIV,SCH MED,HOWARD HUGHES MED INST,DEPT GENET,200 LONGWOOD AVE/BOSTON//MA/02115
Journal: JOURNAL OF EXPERIMENTAL MEDICINE, 1993, V178, N3 (SEP 1), P 1057-1065
ISSN: 0022-1007
Language: ENGLISH Document Type: ARTICLE
Abstract: IP-10 is a member of the -C-X-C- chemokine superfamily of proinflammatory cytokines whose secretion is induced by interferon gamma (IFN-gamma) and lipopolysaccharide (LPS). To date no function has been described for IP-10. We have genetically engineered tumor cells to secrete high levels of murine IP-10 and demonstrate that while IP-10 has no effect on the growth of these tumor cells in culture, it elicits a powerful host-mediated antitumor effect in vivo. The IP-10 antitumor response is T lymphocyte dependent, non-cell autonomous, and appears to be mediated by the recruitment of an inflammatory infiltrate composed of lymphocytes, neutrophils, and monocytes. These results document an important biologic property of IP-10 and raise the possibility that some of the T cell-directed effects of IFN-gamma and LPS may be mediated by this chemokine.

2/7/26 (Item 11 from file: 34)
DIALOG(R)File 34:SciSearch(R) Cited Ref Sci

02467293 Genuine Article#: LD660 Number of References: 20
Title: RECOMBINANT HUMAN INTERFERON-INDUCIBLE PROTEIN-10 IS A CHEMOATTRACTANT FOR HUMAN MONOCYTES AND T-LYMPHOCYTES AND PROMOTES T-CELL ADHESION TO ENDOTHELIAL-CELLS
Author(s): TAUB DD; LLOYD AR; CONLON K; WANG JM; ORTALDO JR; HARADA A; MATSUSHIMA K; KELVIN DJ; OPPENHEIM JJ
Corporate Source: NCI,FREDERICK CANC RES & DEV CTR,MOLEC IMMUNOREGULAT LAB,BIOL RESPONSE MODIFIERS PROGRAM/FREDERICK//MD/21702; NCI,FREDERICK CANC RES & DEV CTR,EXPTL IMMUNOL LAB,BIOL RESPONSE MODIFIERS PROGRAM/FREDERICK//MD/21702; KANAZAWA UNIV,INST CANC RES,DEPT PHARMACOL/KANAZAWA/ISHIKAWA 920/JAPAN/
Journal: JOURNAL OF EXPERIMENTAL MEDICINE, 1993, V177, N6 (JUN 1), P 1809-1814
ISSN: 0022-1007
Language: ENGLISH Document Type: ARTICLE
Abstract: The human cytokine interferon-inducible protein 10 (IP-10) is a small glycoprotein secreted by activated T cells, monocytes, endothelial cells, and keratinocytes, and is structurally related to a family of chemotactic cytokines called chemokines. Although this protein is present in sites of delayed-type hypersensitivity reactions and lepromatous leprosy lesions, the biological activity of IP-10 remains unknown. We report here that recombinant human IP-10 stimulated significant in vitro chemotaxis of human peripheral blood monocytes but not neutrophils. Recombinant human IP-10 also stimulated chemotaxis of stimulated, but not unstimulated, human peripheral blood T lymphocytes. Phenotypic analysis of the stimulated T cell population responsive to IP-10 demonstrated that stimulated CD4+ and CD29+ T cells migrated in response to IP-10. This resembles the biological activity of the previously described T cell chemoattractant RANTES. Using an endothelial cell adhesion assay, we demonstrated that stimulated T cells pretreated with optimal doses of IP-10 exhibited a greatly enhanced ability to bind to an interleukin 1-treated endothelial cell monolayer. These results demonstrate that the IP-10 gene encodes for an inflammatory mediator that specifically stimulates the directional migration of T cells and monocytes as well as potentiates T cell adhesion to endothelium.

2/7/27 (Item 12 from file: 34)
DIALOG(R)File 34:SciSearch(R) Cited Ref Sci

02389075 Genuine Article#: KX816 Number of References: 39
Title: PLATELET FACTOR-IV BINDS TO INTERLEUKIN-8 RECEPTORS AND ACTIVATES NEUTROPHILS WHEN ITS N-TERMINUS IS MODIFIED WITH GLU-LEU-ARG
Author(s): CLARKLEWIS I; DEWALD B; GEISER T; MOSER B; BAGGIOLINI M
Corporate Source: UNIV BRITISH COLUMBIA,BIOMED RES CTR/VANCOUVER V6T 1Z3/BC/CANADA/; UNIV BERN,THEODOR KOCHER INST/CH-3000 BERN 9//SWITZERLAND/
Journal: PROCEEDINGS OF THE NATIONAL ACADEMY OF SCIENCES OF THE UNITED STATES OF AMERICA, 1993, V90, N8 (APR 15), P3574-3577
ISSN: 0027-8424
Language: ENGLISH Document Type: ARTICLE
Abstract: Amino acid deletion and mutagenesis experiments have indicated that the sequence Glu-Leu-Arg (ELR) preceding the first cysteine at the N terminus of interleukin 8 (IL-8) is required for receptor binding and neutrophil activation. Platelet factor 4 (PF4) is structurally related to IL-8 (35% sequence identity) but lacks the N-terminal ELR sequence and comparable effects on neutrophils. We introduced the ELR sequence at the N terminus of PF4 and found that the modified protein was a potent neutrophil activator and attractant. On the other hand, when the ELR sequence was introduced into the corresponding positions of two other proteins related to IL-8, gamma-interferon-inducible protein %%%IP10%%% and monocyte chemoattractant protein 1, neither of them acquired neutrophil-activating properties, indicating that besides ELR additional structural determinants of IL-8 and PF4 are important for binding to IL-8 receptors. The conservation of these binding determinants suggests that PF4 may have evolved from a neutrophil activating protein.

2/7/28 (Item 13 from file: 34)
DIALOG(R)File 34:SciSearch(R) Cited Ref Sci

02189434 Genuine Article#: KJ095 Number of References: 35
Title: IDENTIFICATION OF A NOVEL GRANULOCYTE CHEMOTACTIC PROTEIN (GCP-2) FROM HUMAN TUMOR-CELLS - INVITRO AND INVIVO COMPARISON WITH NATURAL FORMS OF GRP, IP-10, AND IL-8
Author(s): PROOST P; DEWOLFPEETERS C; CONINGS R; OPDENAKKER G; BILLIAU A; VANDAMME J
Corporate Source: CATHOLIC UNIV LEUVEN,REGA INST MED RES,MINDER BROEDERSSTR 10/B-3000 LOUVAIN//BELGIUM/; CATHOLIC UNIV LEUVEN,REGA INST MED RES,MINDER BROEDERSSTR 10/B-3000 LOUVAIN//BELGIUM/; CATHOLIC UNIV LEUVEN,HISTO & CYTOCHEM LAB/B-3000 LOUVAIN//BELGIUM/
Journal: JOURNAL OF IMMUNOLOGY, 1993, V150, N3 (FEB 1), P1000-1010
ISSN: 0022-1767
Language: ENGLISH Document Type: ARTICLE
Abstract: Stimulated human osteosarcoma cells (MG-63) were used as a source of granulocyte chemotactic protein (GCP). In addition to the previously isolated GCP-1/IL-8, natural forms of GROalpha, GROgamma, and IP-10 were purified and identified by amino acid sequence analysis. Further, a novel GCP, GCP-2, was isolated in its natural form (6 kDa) and was

found to be structurally related to the other members of the IL-8 family. GROalpha, IP-10, and GCP-2 showed heterogeneity, in that several forms of each protein were recovered. These differed in truncation at the amino terminus. Reverse phase HPLC allowed us to separate four such different forms of GCP-2. These tumor-derived factors were compared in granulocyte activation and chemotaxis assays. IL-8 induced neutrophil gelatinase B release at 2 nM, but GROalpha and GCP-2 showed a 5- to 10-fold lower specific activity. When the migration of granulocytes through polycarbonate micropore membranes was measured, GCP-2 and GROalpha had a maximal chemotactic index comparable to that of IL-8. The minimal effective dose for GCP-2 and GROalpha was 3 to 10 nM, whereas the specific activity of IL-8 was at least 10-fold higher. IP-10 was not active in this assay at doses up to 100 nM. Finally, in vivo chemotaxis was measured by using granulocyte recruitment in the rabbit skin model. After intradermal injection of 200 ng/site, GCP-2 provoked a significant granulocyte infiltration, albeit to a lesser extent than did IL-8 and GROalpha. GCP-2 did not attract monocytes in vivo nor did it induce the cells in vitro to migrate or to produce enzyme. In conclusion, this study reveals a new member of the IL-8 family and shows that these related inflammatory mediators possess different potencies and efficacies towards granulocytes.

2/7/29 (Item 1 from file: 71)
DIALOG(R)File 71:ELSEVIER BIOBASE

03836445 2007253417
Maternal serum concentrations of the chemokine CXCL10/IP-10 are elevated in acute pyelonephritis during %%%pregnancy%%%
Gotsch F.; Romero R.; Espinoza J.; Kusanovic J.P.; Mazaki-Tovi S.; Erez O.; Than N.G.; Edwin S.; Mazor M.; Yoon B.H.; Hassan S.S.
ADDRESS: Dr. R. Romero, Perinatology Research Branch, NICHD/NIH/DHHS, Wayne State University/Hutzel Women's Hospital, 3990 John R., Detroit, MI 48201, United States
EMAIL: nichdprbchiefstaff@mail.nih.gov
Journal: Journal of Maternal-Fetal and Neonatal Medicine, 20/10 (735-744), 2007, United Kingdom
CODEN: JMNMA
ISSN: 1476-7058 eISSN: 1476-4954
PUBLISHER ITEM IDENTIFIER: 781051544
DOCUMENT TYPE: Article
LANGUAGES: English SUMMARY LANGUAGES: English
NO. OF REFERENCES: 119

Objective. Acute pyelonephritis is one of the most frequent medical complications of %%%pregnancy%%%, as well as a common cause of antepartum hospitalization. Interferon (IFN)-gamma inducible protein, CXCL10/IP-10, is a member of the CXC chemokine family with pro-inflammatory and anti-angiogenic properties. The purpose of this study was to determine whether maternal serum concentrations of CXCL10/IP-10 change in patients with acute pyelonephritis during %%%pregnancy%%%. Study design. This cross-sectional study was conducted to determine the difference in maternal serum concentrations of CXCL10/IP-10 in %%%pregnant%%% women with acute pyelonephritis (N = 41) and normal %%%pregnant%%% women (N = 89). Pyelonephritis was defined in the presence of a positive urine culture, fever, and maternal clinical signs; blood cultures were performed in 36 cases. Maternal serum concentrations of CXCL10/IP-10 were measured by a sensitive immunoassay. Non-parametric statistics were used for analysis. Results. (1) The median serum concentration of CXCL10/IP-10 in %%%pregnant%%% patients with pyelonephritis was significantly higher than in normal %%%pregnant%%% women (median 318.5 pg/mL, range 78.8-2459.2 vs. median 116.1 pg/mL, range 40.7-1314.3, respectively; $p < 0.001$); (2) maternal median serum concentrations of CXCL10/IP-10 did not differ significantly among patients with acute pyelonephritis with and without bacteremia (positive blood cultures: median 362.6 pg/mL, range 100.2-2459.2 vs. negative blood cultures: median 298.9 pg/mL, range 108.5-1148.7, respectively; $p = 0.3$). Conclusions. Pyelonephritis in %%%pregnant%%% women is associated with an increased maternal serum concentration of the chemokine CXCL10/IP-10. (c) 2007 Informa UK Ltd.

2/7/30 (Item 1 from file: 399)
DIALOG(R)File 399:CA SEARCH(R)

146293997 CA: 146(15)293997p JOURNAL
Immunological biomarkers for human papillomavirus infection and early stage of cervical cancer
AUTHOR(S): Sasagawa, Toshiyuki
LOCATION: Graduate School of Medical Science, Division of Health Sciences, Kanazawa University, Kanazawa, Japan,
JOURNAL: Nippon Sanka Fujinka Gakkai Zasshi (Nippon Sanka Fujinka Gakkai Zasshi) DATE: 2006 VOLUME: 58 NUMBER: 11 PAGES: 1745-1751 CODEN: NISFAY ISSN: 0300-9165 LANGUAGE: Japanese PUBLISHER: Nippon Sanka Fujinka Gakkai
SECTION:
CA215008 Immunochemistry
CA214XXX Mammalian Pathological Biochemistry
IDENTIFIERS: IP10 biomarker papillomavirus uterus cervix cancer antibody E2
DESCRIPTORS:
T cell(lymphocyte)...
activation; immunol. biomarkers for human papillomavirus infection and early stage of cervical cancer
Uterus,neoplasm...
cervix; immunol. biomarkers for human papillomavirus infection and early stage of cervical cancer
Proteins...
CIN-2, CIN-3; immunol. biomarkers for human papillomavirus infection and early stage of cervical cancer
Proteins...
E6; immunol. biomarkers for human papillomavirus infection and early stage of cervical cancer
Proteins...
E7; immunol. biomarkers for human papillomavirus infection and early stage of cervical cancer
Interferons...
.gamma.; immunol. biomarkers for human papillomavirus infection and early stage of cervical cancer
Proteins...
gene L1; immunol. biomarkers for human papillomavirus infection and early stage of cervical cancer
T cell(lymphocyte)...
helper cell/inducer, TH1; immunol. biomarkers for human papillomavirus infection and early stage of cervical cancer
T cell(lymphocyte)...
helper cell/inducer, TH2; immunol. biomarkers for human papillomavirus infection and early stage of cervical cancer
Antibodies and Immunoglobulins... Biomarkers... Human papillomavirus 16... Human...
immunol. biomarkers for human papillomavirus infection and early stage of cervical cancer
Chemokines...
interferon .gamma.-inducible protein-10; immunol. biomarkers for human papillomavirus infection and early stage of cervical cancer
Proteins...
MIC2; immunol. biomarkers for human papillomavirus infection and early stage of cervical cancer
Cell activation...
T cell; immunol. biomarkers for human papillomavirus infection and early stage of cervical cancer

2/7/31 (Item 2 from file: 399)
DIALOG(R)File 399:CA SEARCH(R)

144305397 CA: 144(17)305397s JOURNAL

17.beta.-Estradiol suppresses TLR3-induced cytokine and chemokine production in endometrial epithelial cells
AUTHOR(S): Lesmeister, Margaret J.; Jorgenson, Rebecca L.; Young, Steven L.; Misfeldt, Michael L.
LOCATION: Department of Molecular Microbiology and Immunology, School of Medicine, University of Missouri-Columbia, Columbia, MO, USA
JOURNAL: Reprod. Biol. Endocrinol. (Reproductive Biology and Endocrinology) DATE: 2005 VOLUME: 3, PAGES: No pp. given CODEN: RBEEBP
UNIFORM RESOURCE LOCATOR (URL): http://www.rbej.com/content/pdf/1477-7827-3-74.pdf MEDIA TYPE: online computer file ISSN: 1477-7827 LANGUAGE: English PUBLISHER: BioMed Central Ltd.
SECTION:
CA202004 Mammalian Hormones
IDENTIFIERS: estradiol TLR3 cytokine chemokine endometrium epithelium, IL6 IL8 IP10 endometrium epithelium TLR3 estradiol
DESCRIPTORS:
Estrogen receptors...
.alpha.; estrogen receptors-.alpha. of human endometrial epithelium and role in estradiol suppression of TLR3-induced cytokine and chemokine formation
Epithelium...
endometrial; estradiol suppresses TLR3-induced cytokine and chemokine prodn. in human endometrial epithelial cells
Uterus...
endometrium, epithelium; estradiol suppresses TLR3-induced cytokine and chemokine prodn. in human endometrial epithelial cells
Interleukin 6... Interleukin 8...
estradiol modulates TLR3 function through suppression of Poly I:C-induced proinflammatory and antiviral cytokine and chemokine formation by human endometrial epithelium
Cytokines... Chemokines... Human...
estradiol suppresses TLR3-induced cytokine and chemokine prodn. in human endometrial epithelial cells
Reproductive system...
female; steroid hormones effects on TLR3 in regulating immune response of female reproductive system
mRNA...
for TLR-3; estradiol and progesterone effects on TLR-3 mRNA and protein in human endometrial epithelial cells
Chemokines...
interferon .gamma.-inducible protein-10; estradiol modulates TLR3 function through suppression of Poly I:C-induced proinflammatory and antiviral cytokine and chemokine formation by human endometrial e
Immunity...
steroid hormones effects on TLR3 in regulating immune response of female reproductive system
Receptors...
TLR-3 (Toll-like receptor-3); estradiol and progesterone effects on TLR-3 mRNA and protein in human endometrial epithelial cells
CAS REGISTRY NUMBERS:
57-83-0 biological studies, estradiol and progesterone effects on TLR3 expression and function in human endometrial epithelium
50-28-2 biological studies, estradiol suppresses TLR3-induced cytokine and chemokine prodn. in human endometrial epithelial cells
26301-44-0 estradiol modulates TLR3 function through suppression of Poly I:C-induced proinflammatory and antiviral cytokine and chemokine formation by human endometrial epithelium

2/7/32 (Item 3 from file: 399)
DIALOG(R)File 399:CA SEARCH(R)

141117404 CA: 141(8)117404f JOURNAL
Spatial and temporal expression of ligands for CXCR3 and CXCR4 in human endometrium
AUTHOR(S): Kitaya, Kotaro; Nakayama, Takeshi; Daikoku, Nobue; Fushiki, Shinji; Honjo, Hideo
LOCATION: Departments of Obstetrics and Gynecology, Kyoto Prefectural University of Medicine, Kyoto, Japan, 602-8566
JOURNAL: J. Clin. Endocrinol. Metab. (Journal of Clinical Endocrinology and Metabolism) DATE: 2004 VOLUME: 89 NUMBER: 5 PAGES: 2470-2476
CODEN: JCEMAZ ISSN: 0021-972X LANGUAGE: English PUBLISHER: Endocrine Society
SECTION:
CA202004 Mammalian Hormones
IDENTIFIERS: CXCR3 CXCR4 Mig IP10 ITAC SDF1alpha endometrium progesterone estradiol
DESCRIPTORS:
Chemokine receptors...
CXCR3; steroids-induced differential expression of ligands for CXCR3, CXCR4, and recruitment of natural killer cells in human endometrium throughout menstrual cycle
Chemokine receptors...
CXCR4; steroids-induced differential expression of ligands for CXCR3, CXCR4, and recruitment of natural killer cells in human endometrium throughout menstrual cycle
Uterus...
endometrium; steroids-induced differential expression of ligands for CXCR3, CXCR4, and recruitment of natural killer cells in human endometrium throughout menstrual cycle
Chemokines...
I-TAC; steroids-induced differential expression of ligands for CXCR3, CXCR4, and recruitment of natural killer cells in human endometrium throughout menstrual cycle
Chemokines...
interferon .gamma.-inducible protein-10; steroids-induced differential expression of ligands for CXCR3, CXCR4, and recruitment of natural killer cells in human endometrium throughout menstrual cycle
Chemokines...
Mig (monokine induced by interferon-.gamma.); steroids-induced differential expression of ligands for CXCR3, CXCR4, and recruitment of natural killer cells in human endometrium throughout menstrual cy
Lymphocyte...
natural killer cell; steroids-induced differential expression of ligands for CXCR3, CXCR4, and recruitment of natural killer cells in human endometrium throughout menstrual cycle
Chemokines...
SDF-1 (stromal-derived factor-1); steroids-induced differential expression of ligands for CXCR3, CXCR4, and recruitment of natural killer cells in human endometrium throughout menstrual cycle
Cell migration... Human... Ovarian cycle...
steroids-induced differential expression of ligands for CXCR3, CXCR4, and recruitment of natural killer cells in human endometrium throughout menstrual cycle
CAS REGISTRY NUMBERS:
50-28-2 57-83-0 biological studies, steroids-induced differential expression of ligands for CXCR3, CXCR4, and recruitment of natural killer cells in human endometrium throughout menstrual cycle

2/7/33 (Item 4 from file: 399)
DIALOG(R)File 399:CA SEARCH(R)

140247116 CA: 140(16)247116d PATENT
Embryo implantation inhibitor
INVENTOR(AUTHOR): Imakawa, Kazuhiko; Nagaoka, Kentaro; Watanabe, Fumiko
LOCATION: Japan,
ASSIGNEE: Proteinexpress Co., Ltd.
PATENT: PCT International ; WO 200422082 A1 DATE: 20040318
APPLICATION: WO 2003JP11268 (20030903) *JP 2002259268 (20020904)
PAGES: 131 pp. CODEN: PIXXD2 LANGUAGE: Japanese
PATENT CLASSIFICATIONS:
CLASS: A61K-038/00A; A61K-048/00B; A61K-039/395B; A61K-031/7088B; A61P-015/00B; A61P-037/02B; C12N-015/09B; C12Q-001/68B; G01N-033/50B; G01N-033/15B; G01N-033/68B; G01N-033/53B
DESIGNATED COUNTRIES: AE; AG; AL; AM; AT; AU; AZ; BA; BB; BG; BR; BY; BZ; CA; CH; CN; CO; CR; CU; CZ; DE; DK; DM; DZ; EC; EE; ES; FI; GB; GD; GE; GH;

GM; HR; HU; ID; IL; IN; IS; JP; KE; KG; KP; KR; KZ; LC; LK; LR; LS; LT; LU; LV; MA; MD; MG; MK; MN; MW; MX; MZ; NI; NO; NZ; OM; PG; PH; PL; PT; RO; RU; SC; SD; SE; SG; SK; SL; SY; TJ; TM; TN; TR; TT; TZ; UA; UG; US; UZ; VC; VN; YU; ZA; ZM; ZW; AM; AZ; BY; KG; KZ; MD; RU DESIGNATED REGIONAL: GH; GM; KE ; LS; MW; MZ; SD; SL; SZ; TZ; UG; ZM; ZW; AT; BE; BG; CH; CY; CZ; DE; DK; EE; ES; FI; FR; GB; GR; HU; IE; IT; LU; MC; NL; PT; RO; SE; SI; SK; TR; BF; BJ; CF; CG; CI; CM; GA; GN; GQ; GW; ML; MR; NE; SN; TD; TG
SECTION:
CA201012 Pharmacology
IDENTIFIERS: embryo implantation inhibitor interferon gamma inducible protein sequence, pregnancy infertility mol cloning IP10
DESCRIPTORS:
Interferons...
.alpha.; embryo implantation inhibitor
Chemokine receptors...
CXCR3; embryo implantation inhibitor
Fertility...
disorder; embryo implantation inhibitor
Embryo,animal... Pregnancy... Molecular cloning... Sheep... Sheep...
embryo implantation inhibitor
Interferons...
.gamma.; embryo implantation inhibitor
Chemokines...
interferon .gamma.-inducible protein-10, analog; embryo implantation inhibitor
cDNA...
IP-10; embryo implantation inhibitor
Antibodies and Immunoglobulins...
monoclonal, anti-IP-10; embryo implantation inhibitor
Interferons...
.tau.; embryo implantation inhibitor
CAS REGISTRY NUMBERS:
671527-31-4 amino acid sequence; embryo implantation inhibitor
671527-30-3 nucleotide sequence; embryo implantation inhibitor
671527-66-5 671527-67-6 671527-68-7 671527-69-8 671527-70-1 671527-71-2 671527-72-3 671527-73-4 671527-74-5 671527-75-6 671527-76-7 671527-77-8 671527-78-9 671527-79-0 671527-80-3 671527-81-4 671527-82-5 671527-83-6 671527-84-7 671527-85-8 671527-86-9 671527-87-0 unclaimed nucleotide sequence; embryo implantation inhibitor
671527-88-1 671527-89-2 671527-90-5 unclaimed sequence; embryo implantation inhibitor

2/7/34 (Item 5 from file: 399)
DIALOG(R)File 399:CA SEARCH(R)

138253410 CA: 138(17)253410v JOURNAL
A chemokine, interferon (IFN)-.gamma.-inducible protein 10 kDa, is stimulated by IFN-.tau. and recruits immune cells in the ovine endometrium
AUTHOR(S): Nagaoka, Kentaro; Sakai, Akiharu; Nojima, Hisashi; Suda, Yoshihito; Yokomizo, Yuichi; Imakawa, Kazuhiko; Sakai, Senkiti; Christenson, Ronald K.
LOCATION: Laboratory of Animal Breeding, Faculty of Agriculture, University of Tokyo, Tokyo, Japan, 113-8657
JOURNAL: Biol. Reprod. (Biology of Reproduction) DATE: 2003 VOLUME: 68 NUMBER: 4 PAGES: 1413-1421 CODEN: BIREBV ISSN: 0006-3363 LANGUAGE: English PUBLISHER: Society for the Study of Reproduction
SECTION:
CA215005 Immunochemistry
IDENTIFIERS: interferon IP10 leukocyte recruitment chemotaxis pregnancy
DESCRIPTORS:
Interferons...
.alpha.; chemokine, IP 10 is stimulated by IFN-.tau. and recruits immune cells in ovine endometrium
Pregnancy... Embryo,animal... Chemotaxis...
chemokine, IP 10 is stimulated by IFN-.tau. and recruits immune cells in ovine endometrium
Interferons...
.gamma.; chemokine, IP 10 is stimulated by IFN-.tau. and recruits immune cells in ovine endometrium
Chemokines...
interferon .gamma.-inducible protein-10; chemokine, IP 10 is stimulated by IFN-.tau. and recruits immune cells in ovine endometrium
Cell migration...
leukocyte recruitment; chemokine, IP 10 is stimulated by IFN-.tau. and recruits immune cells in ovine endometrium
Interferons...
.tau.; chemokine, IP 10 is stimulated by IFN-.tau. and recruits immune cells in ovine endometrium

2/7/35 (Item 6 from file: 399)
DIALOG(R)File 399:CA SEARCH(R)

137108020 CA: 137(8)108020t JOURNAL
Expression of interferon-.gamma.-inducible protein-10 in human endometrial stromal cells
AUTHOR(S): Kai, Kengo; Nasu, Kaei; Nakamura, Satomi; Fukuda, Junichiro; Nishida, Masakazu; Miyakawa, Isao
LOCATION: Department of Obstetrics and Gynecology, Oita Medical University, Oita, Japan, 879-5593
JOURNAL: Mol. Hum. Reprod. (Molecular Human Reproduction) DATE: 2002 VOLUME: 8 NUMBER: 2 PAGES: 176-180 CODEN: MHREFD ISSN: 1360-9947 LANGUAGE: English PUBLISHER: Oxford University Press
SECTION:
CA215005 Immunochemistry
IDENTIFIERS: cytokine IP10 endometrium interferon interleukin leukocyte
DESCRIPTORS:
Uterus...
endometrium; expression of interferon-.gamma.-inducible protein-10 in human endometrial stromal cells
Human... Interleukin 1.beta.... Lipopolysaccharides... Tumor necrosis factors...
expression of interferon-.gamma.-inducible protein-10 in human endometrial stromal cells
Interferons...
.gamma.; expression of interferon-.gamma.-inducible protein-10 in human endometrial stromal cells
Chemokines...
interferon-inducible protein-10; expression of interferon-.gamma.-inducible protein-10 in human endometrial stromal cells
Leukocyte...
migration; expression of interferon-.gamma.-inducible protein-10 in human endometrial stromal cells

2/7/36 (Item 7 from file: 399)
DIALOG(R)File 399:CA SEARCH(R)

131350279 CA: 131(26)350279x PATENT
Interferon-inducible protein 10 is a potent inhibitor of angiogenesis
INVENTOR(AUTHOR): Tosato, Giovanna; Angiolillo, Anne L.; Sgadari, Cecilia
LOCATION: USA
ASSIGNEE: United States Dept. of Health and Human Services
PATENT: United States ; US 5994292 A DATE: 19991130
APPLICATION: US 455079 (19950531)
PAGES: 29 pp. CODEN: USXXAM LANGUAGE: English
PATENT CLASSIFICATIONS:
CLASS: 514002000; A61K-038/00A
SECTION:
CA215005 Immunochemistry
CA201XXX Pharmacology
CA202XXX Mammalian Hormones
CA214XXX Mammalian Pathological Biochemistry

IDENTIFIERS: IP10 protein angiogenesis inhibition
DESCRIPTORS:
Interferons...
.alpha.; in combination with IP-10 chemokine-derived peptides for angiogenesis inhibition
Immunosuppressants... Neoplasm... Radiotherapy...
anti-angiogenic activity of IP-10 chemokine and its peptide fragments in host immunocompromised by
Antitumor agents...
anti-angiogenic activity of IP-10 chemokine and its peptide fragments in relation to
Lymphoma...
Burkitt's; interferon-inducible protein 10 inhibits neovascularization by
Immunosuppression...
cellular; anti-angiogenic activity of IP-10 chemokine and its peptide fragments in
Nervous system...
central; interferon-inducible protein 10 and its peptide fragments for inhibition of neovascularization in
Skin...
dermis; interferon-inducible protein 10 and its peptide fragments for inhibition of neovascularization in
Eye,disease...
diabetic retinopathy; interferon-inducible protein 10 inhibits neovascularization in
Uterus...
endometrium; interferon-inducible protein 10 and its peptide fragments for inhibition of neovascularization in
Heart...
endothelium; interferon-inducible protein 10 and its peptide fragments for angiogenic factor-induced differentiation of
Blood vessel...
endothelium; interferon-inducible protein 10 inhibits differentiation of
Skin...
epidermis; interferon-inducible protein 10 and its peptide fragments for inhibition of neovascularization in
Drug delivery systems...
for administration of anti-angiogenic IP-10 chemokine and its peptide fragments
Kidney,disease...
Goodpasture's syndrome; interferon-inducible protein 10 and its peptide fragments for inhibition of neovascularization in
Immunodeficiency...
hereditary; anti-angiogenic activity of IP-10 chemokine and its peptide fragments in
Anti-inflammatory agents... Chemotherapy... Thrombospondins...
in combination with IP-10 chemokine-derived peptides for angiogenesis inhibition
Cytokines...
interferon-inducible IP-10; anti-angiogenic activity of native protein and peptide fragments
Atherosclerosis... Autoimmune disease... Bone... Cardiovascular system... Digestive tract... Inflammation... Kidney... Liver... Lymphatic system... Mammary gland... Muscle... Psoriasis... Reproductive tract... Respiratory tract... Rheumatoid arthritis... Sarcoidosis... Sjogren's syndrome... Umbilical cord... Urinary tract...
interferon-inducible protein 10 and its peptide fragments for inhibition of neovascularization in
Hepatocyte growth factor... Interleukin 8...
interferon-inducible protein 10 inhibits angiogenic activity of
Cell differentiation...
interferon-inducible protein 10 inhibits differentiation of vascular endothelium
Wound healing...
interferon-inducible protein 10 inhibits neovascularization in
Sarcoma...
Kaposi's; interferon-inducible protein 10 inhibits neovascularization by
Glaucoma(disease)...
neovascular; interferon-inducible protein 10 and its peptide fragments for inhibition of neovascularization in
Angiogenesis...
neovascularization; peptides of interferon-inducible protein 10 for inhibition of
Peptides,biological studies...
of interferon-inducible protein 10 for angiogenesis inhibition
Angiogenesis inhibitors...
peptides of interferon-inducible protein 10 as
Biliary tract...
primary biliary cirrhosis; interferon-inducible protein 10 and its peptide fragments for inhibition of neovascularization in
Eye...
retina; interferon-inducible protein 10 and its peptide fragments for inhibition of neovascularization in
Eye,disease...
retrolental fibroplasia; interferon-inducible protein 10 and its peptide fragments for inhibition of neovascularization in
Connective tissue...
scleroderma; interferon-inducible protein 10 and its peptide fragments for inhibition of neovascularization in
Liver...
sinusoid, endothelium; interferon-inducible protein 10 and its peptide fragments for angiogenic factor-induced differentiation of
Lupus erythematosus...
systemic; interferon-inducible protein 10 and its peptide fragments for inhibition of neovascularization in
Immunodeficiency...
T-cell deficiency; anti-angiogenic activity of IP-10 chemokine and its peptide fragments in
Thyroid gland,disease...
thyroiditis; interferon-inducible protein 10 and its peptide fragments for inhibition of neovascularization in
Eye,disease...
trachoma; interferon-inducible protein 10 and its peptide fragments for inhibition of neovascularization in
Vein...
umbilical, endothelium; interferon-inducible protein 10 and its peptide fragments for angiogenic factor-induced differentiation of
Blood vessel,disease...
vasculitis; interferon-inducible protein 10 and its peptide fragments for inhibition of neovascularization in
Infection...
viral; anti-angiogenic activity of IP-10 chemokine and its peptide fragments in host immunocompromised by
CAS REGISTRY NUMBERS:
112002-68-3 250345-82-5 250345-83-6 250345-84-7 250345-85-8 250345-86-9 250345-87-0 250345-88-1 250345-89-2 250345-90-5 250345-91-6 250345-92-7 250345-93-8 250345-94-9 250345-95-0 250345-96-1 250345-97-2 250345-98-3 250345-99-4 250346-00-0 250346-01-1 for angiogenesis inhibition
50-24-8 50-78-2 15687-27-1 23110-15-8 37270-94-3 129298-91-5 in combination with IP-10 chemokine-derived peptides for angiogenesis inhibition
106096-92-8 106096-93-9 127464-60-2 interferon-inducible protein 10 inhibits angiogenic activity of
86090-08-6 with interferon-inducible protein 10 for inhibition of angiogenesis
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